

1 Introduction

Our overall aim to develop and implement a model for projecting HIV / AIDS that is simple enough to work with data inputs which are either widely available or can be plausibly substituted by the use of default patterns from other populations. At the same time, the model must be capable of fitting currently available historical time series of HIV prevalence data, and projecting plausible prevalence and incidence trends for at least an equivalent time into the future.

Background

Existing models tend to fall into one of two extremes. The simpler epidemiological models incorporate only the infected population, with new infections generated by an “exogenously” specified new infections curve (as defined in Brookmeyer and Gail 1994:12). Epimodel (Chin and Lwanga 1991) exemplifies models of this type. The models implemented by Tim Brown (1999), which generalize Epimodel by introducing age-specificity and in other ways, are also in this class. More complex epidemiological models, like most demographic models, incorporate the entire population, uninfected as well as infected. They thus make it possible to generate numbers of new infections “endogenously” as a function of numbers of currently infected and uninfected persons, perhaps disaggregated by pertinent characteristics. The differential equation models described in Anderson and May (1991:122-128) and Daley and Gani (1999:20-52) exemplify these models. We refer to the first type as “infected population only” models, the second type as “whole population” models.

Academics have tended to work with the second class of model, developing complex constructions which allow them to investigate the theoretical effects of certain kinds of sexual behavior (e.g. concurrent partnerships) or interventions (e.g. partially effective vaccines). However such models have not been widely used by planners and statisticians in national and international organizations because they tend to be very complicated to set up and run, and because there is little data available as input to generate plausible scenarios. National statistical offices, and organizations in the UN system have worked with the second type of model, because these can be fitted to simple data, such as time series of estimates of numbers of infected persons.

In recent years, there has been some dissatisfaction expressed with the performance of these simple models on two grounds. First, they require strong a priori assumptions about the functional form of the time trend curve describing the epidemic – this has led to a lack of flexibility in extrapolating short term trends to the medium term time horizon required of many demographic projections. Secondly, the fact that they take no account of the size of the population at risk, has led to worries about the plausibility of the large numbers of infections generated in fast growing scenarios – these could conceivably rise to levels incompatible with the number of persons at risk of infection in the population, especially if there were secular trends in fertility tending to decrease the relative size of incoming cohorts.

Objectives

We have adopted three primary research objectives.

- To develop minimally complex whole population models incorporating age, sex and numbers of persons infected and not infected. The essential problem here is to incorporate age-

specificity and endogenous modeling of new infections while maintaining sufficient simplicity for the model to be of use in practical empirical work.

- To explore the extent to which the new infection curves generated by these models recapitulate the various functional forms for the new infection curve assumed by the “infected population only” models. Classical epidemiological models provide some guidance here, but with limited provision for age-specificity given currently available data (Ades and Nokes, 1993; Fontanet et al, 1998).
- To examine the interface between HIV/AIDS models and population projection calculations aimed at describing the long term demographic impact of HIV/AIDS. The driving concern here is to improve on the methods currently in use, which employ ad hoc methods for distributing numbers of infected persons by age and sex and do not adequately address multiple decrement issues. To meet this objective requires a model that incorporates the standard demographic age-sex distribution for a national population and a demographic-epidemiological model that distinguishes AIDS and non-AIDS deaths by age and sex.

Subpopulation at risk

These objectives led to the development of a model in which we mapped the transitions of persons born into the population from a zero risk state into a state where they are at risk of infection. The focus of our empirical work has been heterosexual epidemics in which the at risk state is defined by sexual activity, but the model formulation is general, assuming only that there are age-specific risk functions governing entrance into and departure from the at risk state, e.g., the transition into or out of the state of being a needle-sharing intravenous drug user.

This conceptualization of transitions into and out of an “at risk” state is important for the study of heterosexual epidemics for two reasons. First, it allows us to utilize widely available data on start of sexual activity to give an empirical grounding to our age-specific risk of infection patterns. Second, it gives us the chance to establish a link with fertility patterns, thereby enabling the output of internally consistent levels of vertical transmission, and patterns of HIV prevalence in pregnant women, reflecting the chief source of prevalence data – the sentinel surveillance of women in ante-natal clinics.

The force of infection

The central epidemiological feature of the model is the way in which the force of infection for the uninfected at risk population depends on the number of persons already infected. In our formulation the force of infection has two components, which we term “exogenous” and “endogenous.” The exogenous component is independent of the current prevalence of HIV in the population. It represents the initial arrival of HIV in the heterosexually active population and any form of repeated re-introduction of HIV via “bridging” populations, e.g. from infected blood products. The general model allows this component to change with time in any manner whatever, though our application to Uganda assumes it constant.

The endogenous component is a multiplier that is applied to the current prevalence of HIV in the population to determine the level of new infections resulting from persons already infected. This second, endogenous component has, in turn, two additive components. The first is constant over time and representing a level of risk that cannot be reduced except by universal, life long monogamy. The second may change over time, representing behavioral and biomedical factors that may change in such a way as to reduce the level of risk inherent in any given prevalence,

e.g., extent of condom use, rate of partner change and degree of co-infection with other STDs. This second endogenous multiplier component may decline rapidly in response to the threats posed by the epidemic as it develops and becomes recognized.

Model projections

The model is used to produce projections that show precisely how the principal demographic and epidemiological features of the population change as an epidemic develops and subsides. This change is controlled by the values of the various demographic and epidemiological parameters incorporated in the model, including those governing HIV-related and other mortality, fertility, transition to sexual activity, and infection. In the illustrative application for Uganda the mortality, fertility and sexual transition parameters are all fixed on the basis of direct empirical evidence. The parameters governing the force of infection will in general be fitted indirectly, by initiating the projection process with plausible guesses at their likely values, and then adjusting these so as to minimize the difference between projected and observed prevalence levels in the historical time series available for the given population.

Overview of the report

The following chapter presents a simple model, lacking age and duration specificity, that is used to gain insight about the way in which the force of infection is handled in the general model. Chapters 3 and 4 develop, respectively, the general model and a prototype computer program for its implementation. Chapters 5 and 6 present an illustrative application to Uganda, first reviewing and working the available data into the form required by the model and then examining the fit of the model to prevalence data for 1985-1998.